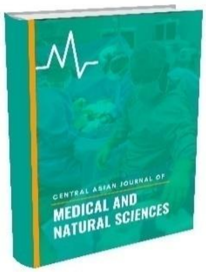
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Renal and Extra-Renal Diseases, Their Etiological and Epidemiological Manifestations

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Abstract: At present, there is a trend towards an increase in kidney diseases due to the influence of such environmental factors as the accumulation of lead in the body, an increase in alcohol consumption, which contribute to the spread of urate dysmetabolism, which has a population character. This determines the relevance of the problem of early diagnosis of metabolic disorders, manifested by kidney damage. Detection of urate nephropathy in the early stages can prevent the development of nephrosclerosis and end-stage renal failure [1].

Key words: Epidemiological, Diseases, kidney diseases.

One of the most common causes of renal failure, according to a number of studies, is acute alcohol intoxication against the background of cardiovascular diseases (58%), hemorrhagic fever with renal syndrome, poisoning with alcohol surrogates, mushrooms and household poisons. In patients with acute renal failure, pronounced violations of the excretory function of the kidneys with a plasma creatinine (Cr) concentration (Pcr) over 0.4-0.5 mmol were registered. In hemorrhagic fever with renal syndrome, this figure reached 0.9 mmol/l.

Uncontrolled intake of analgesics is also a risk of developing nephropathy and renal failure.

Analgesic nephropathy is a common pathology in many countries of the world. Its frequency in Sweden is 18.1%, in Belgium - 11.8%, in Germany - 6%, in the Czech Republic - 4%, in the USA and Great Britain - from 7-13% [3].

Analgesic nephropathy leads to the development of end-stage renal failure, and among dialysis patients it is detected, on average, in 17.9% of cases.

Diagnosis of the early stage of urate nephropathy is more reliable with a family approach in the interpretation of purine metabolism disorders [5].

Ischemic nephropathy is widespread among elderly and senile patients, often remaining undiagnosed. Ischemic nephropathy is one of the leading causes of impaired renal function. Among patients older than 50 years with end-stage renal disease, ischemic nephropathy is 15% [7].

There is currently no generally accepted classification of ischemic kidney disease. More often, ischemic nephropathy is classified according to 2 criteria:

- the rate of progression of renal failure with the release of acute (rapidly progressive) and chronic;

- localization of lesions of the renal arteries (large, medium and small caliber).

The most common cause of ischemic kidney disease is atherosclerosis, which made it possible to isolate the atherosclerotic form of coronary kidney disease.

Acute renal failure is caused by a sudden violation of the patency of the vessels of the kidneys and the impoverishment of the renal blood flow. This is evidenced by a sharp increase in blood pressure, leukocytosis, fever, intense pain in the kidneys or back. The course of acute renal failure is usually asymptomatic.

Blockage of blood vessels by cholesterol crystals is the main cause of the development of rapidly progressive renal failure in ischemic nephropathy. In addition to the kidneys, the skin, nervous system, and gastrointestinal tract are involved in the pathological process. Signs of this type of kidney failure are; increased blood creatinine; blood pressure; pain, nausea, vomiting, gastrointestinal bleeding, toe necrosis, erythema nodosum and others. Blockage of blood vessels by cholesterol crystals is confirmed by their detection in particles of the skin, muscles, internal organs, primarily the kidneys, liver, pancreas

Torpid (sluggish) renal failure is the most common clinical variant of ischemic nephropathy. As a rule, it is diagnosed only by special functional studies of blood vessels (arteriography, duplex ultrasonography) or by autopsy. It is important to remember that the likelihood of renal vascular disease is high in elderly patients with generalized atherosclerosis.

Given that often ischemic nephropathy is asymptomatic, with signs of renal failure in elderly patients, it is advisable to suspect the presence of this pathology.

Ischemic nephropathy is possible in elderly patients with an unexplained increase in creatinine (more than 30-40% of baseline values), the development of acute renal failure after the appointment of ACE inhibitors; with the development of pulmonary edema against the background of poorly controlled arterial hypertension and chronic renal failure; in the presence of refractory arterial hypertension; with the development of arterial hypertension and azotemia (increased levels of nitrogenous products in the blood) in a patient with a transplanted kidney; when a progressive azotemia is detected in a patient with vascular lesions (coronary arteries, vessels of the brain, lower extremities, aneurysms of the abdominal aorta, etc.); in the presence of noise in the projection of the renal arteries; with prolonged smoking.

In these clinical situations that are dangerous for the development of ischemic nephropathy, additional instrumental studies are needed for diagnostic purposes: duplex ultrasonography, radioisotope renography, nuclear magnetic tomography, and the specificity of these methods ranges from 83 to 100%.

In recent years, computed angiography has been used to diagnose ischemic nephropathy [4,5].

According to data [8,9], the progression of nephropathies depends on pathogenetic and nosological characteristics: immune and infectious-inflammatory, with a predominant primary lesion of the glomeruli or tubulo-interstitial apparatus; metabolic, vascular. Prognostic value have gender, age of the patient, heredity, smoking, obesity; the presence of foci of infection, chronic inflammatory diseases of the genital organs caused by bacteria, chlamydia; allergic reactions, viral infections. Syndromic characteristics of nephropathies are of great importance: acceleration of progression in nephrotic syndrome; stable hypertensive syndrome in chronic renoparenchymal pathological conditions, which determines their staging and worsens the prognosis.

When diagnosing nephropathy, it is important to take into account laboratory test data: the degree and selectivity of proteinuria, infection of the urinary system, fibronectin excretion, transamidase activity

of urine, hyperuricemia, hyperuricosuria, lipid metabolism, immune homeostasis. The results of prognostic tests of nephropathy should be taken into account when developing standards for examining patients at the stages of nephrological care.

Epidemiological studies are currently underway on the prevalence of kidney disease in different regions of the world. According to their data, for example, this figure reaches 13%. in the United States, which is 26 million people. Moreover, most patients belong to the first three stages of chronic kidney disease.

According to researchers from Russia, the prevalence of early stages of chronic kidney disease was 36%.

A joint team of Israeli and American scientists has discovered a piece of DNA containing a genetic risk factor for kidney disease in humans. This is a new region of the MYH9 gene, the presence of which explains the fatal kidney disease why some people develop it more often [3].

In the United States, there are 40 million patients with chronic kidney disease of varying severity. Half a million of them are diagnosed at an early stage of kidney disease, for which there is no cure today. There are about 5,000 such patients in Israel. These people are offered life-saving measures such as dialysis or a kidney transplant. They can prolong life, but do not provide any satisfactory quality of life. Mortality among these patients remains high.

It has recently been established that genetic factors influence the manifestation of kidney disease. Studies conducted in the United States have shown that the influence of genetics on people from African countries is 4 times greater compared to Europeans. Among Europeans, Spanish genes are twice as risky. In addition, it was found that the socio-economic characteristics of life do not explain significant differences in the incidence of fatal kidney diseases among different population groups. A region on chromosome 22, called the MYH9 gene, has been discovered in which changes appear that explain the incidence of chronic kidney disease in Africans.

Israeli experts conducted a study on immigrants from Ethiopia living in Israel and found that the incidence of chronic kidney disease in them does not exceed the national average.

Recent discoveries in the field of medical genetics help in the development of methods for the early diagnosis of chronic kidney diseases and methods for their preventive treatment in order to prevent the transition of the disease to an irreversible stage.

The tubulointerstitial component is morphologically a combination of diffuse hyaline and hyaline-hydropic dystrophy of the tubular epithelium and changes in the renal interstitium in the form of cellular infiltrates and sclerosis.

Violations of the morphofunctional state of the tubules and interstitium underlie tubular dysfunctions. Fibrosis of the renal interstitium is considered as a sign of the progression of primary interstitial renal lesions - chronic pyelonephritis, interstitial nephritis. Parameters reflecting the severity of tubulointerstitial changes have prognostic value in various forms of chronic glomerulonephritis in children and adults with the progression of CKD.

That pathological changes in the glomeruli determine the progression of chronic renal failure in diseases with the primary involvement of these structures and that tubulointerstitial changes only accompany them. Currently, there are more and more works showing that in glomerular diseases, a decrease in the level of glomerular filtration correlates mainly with the degree of tubulointerstitial rather than glomerular damage, and most of the events that determine the outcome of these diseases occur in the interstitium [7].

Proximal tubular cells are a powerful additional source of platelet growth factor. Endothelin 1, secreted by the same cells, in addition to the monocytic chemoattractant effect, stimulates interstitial fibroblast proliferation and extracellular matrix synthesis.

Proximal tubular cells can themselves produce such extracellular matrix proteins as interstitial collagen types 1 and 3 [2].

In recent experimental studies, another pathway for the development of interstitial fibrosis has been discovered - the transformation of tubular epithelial cells into myofibroblasts under the dose-dependent effect of transforming growth factor.

The amount of extracellular matrix in the interstitium is determined by the ratio of protein production and degradation by proteinases. In this regard, interstitium fibrosis may be a consequence of a decrease in the activity of proteinases, possibly due to an imbalance in the proteinase-inhibitor. Under experimental conditions, an increase in the expression of inhibitors such as; metalloproteinases, TIMP 1, 2 in the presence of pronounced fibrotic changes, although the source of these inhibitors has not been reliably established.

Accumulation of extracellular matrix protein increases the gap between tubular cells and capillaries, reducing oxygen delivery to the tubules. In combination with high metabolic activity, this increases the sensitivity of tubular cells to ischemic effects, the development of which is associated with damage to glomerular vessels in glomerular diseases, which is expressed in a decrease in post-glomerular blood supply to the interstitium. Increased production of endothelin-1 may also be the cause of local vasoconstriction and a decrease in interstitial blood flow [8].

It should be taken into account that C5b-9 membrane-attacking complex of complement components, transferrin due to iron as a cause of peroxidation in proximal tubular cells, has a cytotoxic effect directed at proximal tubular cells. High proteinuria leads to the rupture of lysosomes necessary for increased protein metabolism, which manifests itself in the release of enzymes into the cytoplasm with subsequent cellular damage.

Protein overload activates proximal tubular cells to regulate the production of endothelial chemoattractant factors.

These mechanisms of damage to tubular cells take place both at the beginning of the development of tubulointerstitial changes, and later, aggravated as the tubulointerstitial component forms and ultimately leads to atrophy of the tubular epithelium.

Scientific data on the role of tubulointerstitial changes in the progression of renal disease expand the understanding of the pathogenetic mechanisms of chronic renal disease. Recent experimental and clinical studies have confirmed the influence of immune and non-immune mechanisms on the formation of the tubulointerstitial component. Further study of this process is needed to improve the diagnosis (urinary excretion of cytokines and growth factors) and treatment of chronic kidney disease.

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